

How to Cite:

Al-Jasser, S. A., E Alharbi, S. S., Albishri, B. A., Ghassap, S. A., Rashad, A. I., Alanizi, A. S., AL Khathami, M. M. M., Alareefi, H. S., Brahim Alshammari, H. M., Abdullah Alkhathami, B. M., Dobayan Alenazi, N. S., Mohammed Alhazmi, F. K., Isa Gaddourah, A. M., & Alrakhimy, H. D. (2020). EMS and paramedic management of respiratory distress: Prehospital use of CPAP and BiPAP. *International Journal of Health Sciences*, 4(S1), 269–291.

<https://doi.org/10.53730/ijhs.v4nS1.15219>

EMS and paramedic management of respiratory distress: Prehospital use of CPAP and BiPAP

Saleh AbdulLatif Al-Jasser

KSA, National Guard Health Affairs

Sultan Sulaiman E Alharbi

KSA, National Guard Health Affairs

Abdullah Atiyyan Albishri

KSA, National Guard Health Affairs

Sawsan Ahmad Ghassap

KSA, National Guard Health Affairs

Alaa Ibrahim Rashad

KSA, National Guard Health Affairs

Anoud Saud Alanizi

KSA, National Guard Health Affairs

Mohammed Mesfer Musaed AL Khathami

KSA, National Guard Health Affairs

Hind Saad Alareefi

KSA, National Guard Health Affairs

Hassan Mohammed Brahim Alshammari

KSA, National Guard Health Affairs

Bandar Mohammad Abdullah Alkhathami

KSA, National Guard Health Affairs

Nawaf Subhi Dobayan Alenazi

KSA, National Guard Health Affairs

Fares Khalid Mohammed Alhazmi

KSA, National Guard Health Affairs

Ahmad Mohammed Isa Gaddourah
KSA, National Guard Health Affairs

Hamad Dafalh Alrakhimy
KSA, National Guard Health Affairs

Abstract---Background: Acute Respiratory Distress Syndrome (ARDS), first identified in the 1960s, manifests as acute hypoxic respiratory failure due to diverse causes like infection and trauma. The incidence varies globally, affecting 7.2 to 34 per 100,000 person- years. While ARDS's historical mortality rate was around 60%, advancements in critical care have reduced it to 26-35%. Despite improvements, ARDS accounts for approximately 75,000 U.S. deaths annually and 3 million global cases, contributing significantly to ICU admissions and mechanical ventilation needs. **Aim:** This article aims to explore the prehospital management of respiratory distress in ARDS patients, focusing on the effectiveness of Continuous Positive Airway Pressure (CPAP) and Bilevel Positive Airway Pressure (BiPAP) in the emergency medical services (EMS) setting. The review focus also on radiological picture of ARDS. **Methods:** A comprehensive review of existing literature was conducted, analyzing studies on CPAP and BiPAP application in ARDS management prehospital settings. The review encompasses efficacy, clinical outcomes, and safety of these non-invasive ventilation strategies. **Results:** Evidence indicates that both CPAP and BiPAP are beneficial in improving oxygenation and reducing the need for intubation in ARDS patients. These interventions also enhance patient comfort and can stabilize conditions during transport to definitive care. **Conclusion:** Prehospital use of CPAP and BiPAP presents a promising approach for managing respiratory distress in ARDS. Incorporating these non-invasive ventilation methods can potentially reduce morbidity and mortality, highlighting the need for further training and protocols in EMS systems.

Keywords---ARDS, CPAP, BiPAP, respiratory distress, emergency medical services, non-invasive ventilation.

Introduction

Acute respiratory distress syndrome (ARDS) was initially identified by Ashbaugh et al. in the 1960s as the occurrence of acute hypoxic respiratory failure in adults, triggered by various underlying conditions such as infection, trauma, or pancreatitis. These events result in pulmonary inflammation and nonhydrostatic pulmonary edema [1]. Global estimates suggest that the incidence of ARDS ranges from 7.2 to 34 cases per 100,000 person-years [2-4]. Historically, the case fatality rate of ARDS was approximately 60% [5-7]. However, over the past two decades, there has been a marked improvement in ARDS survival, with current mortality rates reported between 26% and 35% [5-7]. This improvement is largely

attributed to advancements in critical care, particularly the use of low tidal volume mechanical ventilation in managing ARDS patients. Despite these advances, ARDS remains a fatal condition, causing approximately 75,000 deaths annually in the United States [7]. Globally, ARDS impacts around 3 million people each year, accounting for 10% of intensive care unit (ICU) admissions and 23% of ICU patients requiring mechanical ventilation [8].

The clinical definition of ARDS was first established in 1994 by the American- European Consensus Conference (AECC) [9] and later updated in 2012 by the Berlin definition [10,11]. The AECC established criteria for both ARDS and acute lung injury (ALI), with ARDS being a more severe form of hypoxia compared to ALI[9]. The Berlin definition eliminated the ALI category and instead classified ARDS into three severity categories: mild ($200 \text{ mm Hg} < \text{PaO}_2/\text{FiO}_2 \leq 300 \text{ mm Hg}$), moderate ($100 \text{ mm Hg} < \text{PaO}_2/\text{FiO}_2 \leq 200 \text{ mm Hg}$), and severe ($\text{PaO}_2/\text{FiO}_2 \leq 100 \text{ mm Hg}$) [11]. The Berlin definition retains the general diagnostic criteria, which emphasize the acute onset of the condition (within one week), the presence of diffuse bilateral opacities on chest radiographs, and the exclusion of congestive heart failure or intravascular volume overload as the cause. Furthermore, patients must receive a minimum positive end-expiratory pressure (PEEP) of 5 cm H₂O, delivered invasively or non-invasively depending on disease severity. The Berlin definition has shown greater predictive validity for mortality in ARDS patients compared to the AECC definition.

The pathophysiology of ARDS is characterized by diffuse alveolar damage (DAD) and increased capillary permeability [12,13]. Both the capillary endothelial and alveolar epithelial surfaces are compromised, leading to the disruption of the alveolar-capillary membrane. This disruption results in the leakage of protein-rich fluid, the infiltration of neutrophils and macrophages into the alveolar space, and the formation of hyaline membranes [13-16]. The ongoing lung injury and inflammatory response are driven by cytokine activation and the release of pro-inflammatory mediators, including tumor necrosis factor and interleukins IL-1 and IL-6 [12,17]. Activated neutrophils release toxic substances that cause oxidative damage to cells [17]. The damage to the alveolar-capillary membrane causes the accumulation of protein-rich fluid in the pulmonary interstitium, surfactant inactivation, atelectasis, and impaired gas exchange [13,17]. Clinically, this early exudative phase of ARDS is marked by severe hypoxemia and decreased lung compliance [3]. The acute phase may resolve or progress into a fibroproliferative phase characterized by persistent hypoxemia, increased dead space, further loss of lung compliance, lung fibrosis, and neovascularization [17].

The diagnostic criteria for ARDS do not require a histopathological diagnosis of DAD, and the correlation between the clinical diagnosis of ARDS and pathological findings of DAD is variable. Studies comparing clinical ARDS criteria with postmortem, or biopsy evidence of DAD have shown a 50% to 88% correlation using the AECC definition [18-21], and a 45% to 56% correlation with the Berlin definition [22]. Clinicians should be aware of the potential discordance between ARDS diagnosed clinically and the pathological findings of DAD in lung biopsies or autopsies, though the clinical implications of this observation may be limited in most practice settings [23].

Etiology and Risk Factors

Among the more than 50 disorders linked to the onset of ARDS, sepsis, pneumonia, aspiration, trauma, and multiple blood transfusions are responsible for the majority of cases [24,25]. Notably, approximately 20% of ARDS cases lack identifiable risk factors [26]. While a genetic predisposition to the development and severity of ARDS has been proposed, no definitive genetic link has been established [27–30]. Sepsis is the leading cause of ARDS, accounting for around 40% of cases [24,25]. Between 6% to 7% of sepsis patients develop ARDS, with lower rates observed in non-pulmonary causes or less severe forms of sepsis, and higher rates with worse outcomes in those with septic shock [31–34]. Sepsis from a pulmonary origin poses a greater risk for ARDS due to both direct local inflammation and indirect systemic inflammatory responses [7,35,36]. Pneumonia is another frequent cause of ARDS, particularly in hospitalized patients diagnosed with culture-positive pneumonia. Both Gram-positive and Gram-negative bacteria contribute to ARDS at similar rates [37]. While viral and fungal pathogens are less common, they pose a higher risk of ARDS than bacterial pneumonia, particularly pathogens like *Pneumocystis jiroveci* and *Blastomyces* [37].

Aspiration of gastric contents is a significant contributor to ARDS, responsible for up to 30% of cases in some studies [25,38]. ARDS caused by aspiration tends to be more severe and leads to higher mortality rates (approximately three times higher) compared to ARDS from other causes [38]. Factors that increase the likelihood of ARDS following aspiration include male gender, a history of alcohol abuse, lower Glasgow Coma Scale scores, and nursing home residence [38]. Severe trauma accounts for approximately 25% of ARDS cases [24], with an incidence rate of around 12% among trauma patients admitted to ICUs [39]. Although ARDS in trauma patients is linked to extended ICU stays, it does not necessarily predict higher mortality [39]. After adjusting for age, illness severity, and comorbid conditions, trauma-associated ARDS is associated with better survival compared to ARDS from other causes [39]. For instance, in the ARDS Network study, trauma patients with ARDS had a significantly lower risk-adjusted odds of death at 90 days (odds ratio [OR], 0.44; 95% confidence interval [CI], 0.24-0.82; $P = .01$) compared to patients with ARDS from other etiologies [40]. This survival advantage may be due to less severe injury to the lung epithelium and endothelium in trauma-related ARDS [40,41].

Blood transfusions account for 25% to 40% of ARDS cases [24–26]. Transfusion-related acute lung injury (TRALI) is defined as acute lung injury (ALI) that develops within six hours after the transfusion of one or more plasma-containing or plasma-derived blood products [42]. Early studies on transfusions revealed that massive transfusions—over 22 units of blood within 12 hours and over 15 units within 24 hours—are significant risk factors for ARDS [24–26]. In critically ill patients, transfusion of packed red blood cells (PRBCs) is independently associated with ARDS in a dose-response relationship [43]. Patients receiving fresh-frozen plasma and platelet transfusions are at a higher risk of developing ARDS compared to those receiving only PRBC transfusions [44].

ARDS Scoring Systems

In 2011, the **US Critical Illness and Injury Trials Group** developed and validated the **Lung Injury Prediction Score (LIPS)** to identify patients at high risk of developing acute lung injury (ALI) and ARDS before the onset of injury [32]. This scoring system was tested in a multicenter, observational cohort study of 5584 patients with one or more ARDS risk factors, of which 377 (6.8%) developed ALI/ARDS. Patients were evaluated within the first six hours of initial emergency department evaluation or before high-risk elective surgery. LIPS aims to recognize high-risk patients early, enabling the implementation of interventions to prevent the progression to ALI/ARDS. A LIPS score of >4 was identified as the optimal cutoff, with a **negative predictive value** of 0.97 and a **positive predictive value** of 0.18. Sensitivity and specificity were 69% and 78%, respectively. Despite its potential utility, the low positive predictive value and complexity of the LIPS worksheet limit its broader clinical application.

In another study, **Levitt and colleagues** designed the **Early Acute Lung Injury (EALI) score**, aimed at predicting the progression to positive pressure ventilation in patients with radiographic evidence of ALI [45]. Independent predictors such as tachypnea, immune suppression, and increasing oxygen requirements were incorporated into the 3-component EALI score. An EALI score of ≥ 2 identified patients at high risk for progressing to ARDS and requiring positive pressure ventilation, with a sensitivity of 89% and specificity of 75%. The **median time** to needing ventilation was 20 hours. While promising as a triage tool, the EALI score has yet to be validated in external cohorts.

Diagnostic Biomarkers for ARDS:

Given the limitations of ARDS diagnostic criteria and predictive scoring systems, there is increased interest in identifying **biomarkers**. **Exhaled biomarkers** are particularly attractive as they may more accurately reflect lung-specific events. Investigated exhaled biomarkers include volatile organic compounds, cytokines, hydrogen peroxide, nitric oxide, acidity, lipid peroxidation byproducts, and cytokeratins [46]. However, none are currently ready for clinical use.

Other biomarkers from **bronchial alveolar lavage** and **serum** have shown promise. For example, elevated **IL-8** levels in bronchial alveolar lavage have been associated with a higher risk of developing ARDS in high-risk patients [47]. Additionally, **serum lipopolysaccharide-binding protein** has been shown to predict ARDS development in septic patients and is linked to poorer clinical outcomes [48]. Elevated **serum angiopoietin-2** levels have been associated with increased ALI risk in critically ill patients [49]. A recent systematic review identified 20 serum biomarkers for ARDS diagnosis in high-risk populations [50]. While no single biomarker can predict ARDS progression or outcomes reliably, combining multiple biomarkers with clinical data, such as the **APACHE-III scoring system**, has shown promise in improving risk prediction [51,52]. The future utility of ARDS biomarkers may lie in enhancing scoring systems rather than serving as stand-alone diagnostic tests.

Radiological Picture of ARDS

The radiological picture of **Acute Respiratory Distress Syndrome (ARDS)** is typically characterized by specific findings on a **chest X-ray** or **computed tomography (CT) scan** that reflect the severity and extent of lung damage. The key features include:

Chest X-ray (CXR)

1. **Bilateral Opacities:** ARDS is often identified by the presence of diffuse, bilateral, patchy opacities (also called infiltrates) that cover both lungs. These opacities represent the accumulation of fluid, protein, and cells in the alveoli, which causes impaired gas exchange.
2. **Ground-Glass Appearance:** The lungs often exhibit a hazy, "ground-glass" appearance on the X-ray. This indicates partial filling of the alveoli with fluid, leading to reduced air content.
3. **Non-Cardiogenic Pulmonary Edema:** While pulmonary edema can also be seen in heart failure, ARDS-related edema is not caused by heart problems. There is no evidence of cardiomegaly (enlarged heart) or pleural effusions (fluid accumulation around the lungs), which are more common in cardiac-related causes of edema.
4. **Absence of Focal Consolidation:** Unlike pneumonia, where consolidation tends to be more localized, ARDS typically shows diffuse, widespread opacities across both lungs.

Computed Tomography (CT Scan)

1. **Ground-Glass Opacities (GGO):** More clearly visible on CT than on X-ray, GGOs are areas where the alveoli are partially filled with fluid or collapse. This results in hazy, cloud-like regions across the lung fields.
2. **Air Bronchograms:** Air-filled bronchi may be visible against the background of consolidated lung tissue due to fluid-filled alveoli. This is a classic sign of ARDS.
3. **Dependent Consolidation:** CT scans often show denser consolidation in the **dependent areas of the lungs**, particularly in the posterior lower lobes, due to the effects of gravity on fluid accumulation when a patient is in a supine position.
4. **Heterogeneous Distribution:** Unlike some lung diseases that affect the lungs uniformly, ARDS may display heterogeneous patterns of lung involvement, with some areas of the lung appearing relatively normal, while others are severely affected.
5. **Honeycombing or Fibrosis** (in late stages): In chronic or late-stage ARDS, scarring or fibrosis can occur, leading to structural changes in the lung tissue and the formation of small cyst-like spaces that give a honeycomb appearance.

Progression Over Time

- In **early ARDS**, X-rays and CT scans might only show mild ground-glass opacities or subtle bilateral infiltrates.
- As ARDS progresses, the radiographic findings tend to worsen, with increasing infiltrates and consolidation across larger areas of the lung.

- In the **fibrotic phase** (chronic stage), imaging may reveal reduced lung volumes, increased fibrosis, and persistent consolidations.

The radiological findings of ARDS, especially on CT scans, are essential for monitoring the progression of the disease and assessing the response to treatment, though they are not specific and must be interpreted in conjunction with clinical findings.

Management of ARDS

The lung injury seen in **Acute Respiratory Distress Syndrome (ARDS)** is often interpreted as a maladaptive response to an initial insult such as sepsis, pneumonia, or aspiration. However, only a fraction of affected patients will progress to ARDS, leading to significant interest in identifying the early pathophysiological mechanisms that predispose individuals to ARDS, and developing interventions to counteract these injurious processes. Preventative strategies for ARDS, tested in high-risk populations, include early goal-directed therapy in sepsis, intravenous fluid management, blood transfusion protocols, lung-protective ventilation (LPV), and nutritional management [53].

Sepsis Management

Sepsis accounts for nearly 40% of all ARDS cases. While no specific intervention has been proven to completely prevent ARDS in septic patients, delays in sepsis treatment elevate the risk. Specifically, delays in goal-directed resuscitation and timely antibiotic administration increase the odds of ARDS by 3.6- and 2.4-fold, respectively. Early detection and treatment of sepsis significantly reduce ARDS risk [53].

Fluid Management

ARDS is typified by increased capillary permeability and subsequent extravascular lung water accumulation. A **conservative intravenous fluid strategy** has been proposed to reduce ARDS risk in high-risk patients. Research by Jia et al. demonstrated that a high positive fluid balance in mechanically ventilated patients (over 48 hours) was associated with a 1.3-fold increase in ARDS incidence, suggesting that conservative fluid management may be protective [54]. Further, studies in surgical patients have shown that a positive postoperative fluid balance is an independent risk factor for ARDS, with liberal perioperative fluid administration significantly correlating with increased ARDS incidence [55, 56, 57]. Although definitive data on optimal fluid strategies are still pending, excessive IV fluid administration should be avoided in patients at high risk for ARDS [57].

In ARDS patients, the **Fluids and Catheters Treatment Trial (FACTT)** demonstrated that a conservative fluid management approach reduces the duration of mechanical ventilation and ICU length of stay, while improving oxygenation [58]. Negative fluid balance by day 4 was associated with lower hospital mortality and increased ventilator-free and ICU-free days [59]. Thus, controlled diuresis and limiting fluid intake can improve outcomes in ARDS patients [60].

Blood Transfusion Management

There is a dose-dependent relationship between the volume of transfused blood products and ARDS risk, indicating that **restrictive transfusion strategies** may lower ARDS incidence [43, 44, 61]. A Canadian randomized trial found that maintaining a lower target hemoglobin level (7-9 g/dL) significantly reduced ARDS rates compared to a more liberal transfusion strategy (10-12 g/dL) [62]. Caution is particularly advised with transfusion of platelets and fresh-frozen plasma, which have been associated with a higher ARDS risk than red blood cell transfusions [44]. In trauma care, after hemorrhage control, a conservative approach to transfusion has been advocated to mitigate ARDS risk [63].

Granulocyte and HLA-specific antibodies in donor blood have been implicated in the pathogenesis of transfusion-related ARDS through complement activation and pulmonary injury [64, 65]. Screening for these antibodies in donors has been suggested as a potential preventive measure, although the cost-effectiveness and appropriate cutoff levels for screening remain unclear [66]. Concerns about blood safety, particularly regarding plasma and whole blood from female donors with multiple pregnancies, have prompted recommendations from the American Association of Blood Banks to use male donors or females who have never been pregnant or test negative for HLA antibodies [66, 69]. International implementation of these guidelines has led to a significant reduction in TRALI cases [70].

Although some evidence suggests that blood storage time might influence ARDS risk due to neutrophil activation in older blood products, human studies have not consistently supported this association [67, 68, 73]. Consequently, no definitive recommendations regarding the use of newer blood products in high-risk ARDS patients can be made at present [67, 68, 74].

Management of Mechanical Ventilation

Lung-protective ventilation (LPV) strategies represent the most significant advancement in managing ARDS over the past five decades. These strategies, which aim to mitigate volutrauma and atelectrauma through low tidal volumes, reduced inspiratory and plateau pressures, and prone positioning, have demonstrated improved outcomes for ARDS patients. The major critical care societies strongly endorse these LPV strategies in their current ARDS guidelines, which recommend: (1) targeting tidal volumes between 4 to 8 mL/kg (based on predicted body weight), and (2) maintaining plateau pressures below 30 cm H₂O using lower inspiratory pressures [76-78]. Notably, the guidelines discourage routine high-frequency oscillatory ventilation (HFOV) for ARDS patients, as studies like the OSCILLATE trial have linked HFOV to increased 28-day mortality (relative risk 1.41; 95% CI 1.12-1.79) [79]. Other HFOV trials have also failed to demonstrate benefit [80,81]. Further research is needed to determine the safety and efficacy of extracorporeal membrane oxygenation (ECMO) in severe ARDS cases. In a recent randomized controlled trial, ECMO did not significantly reduce mortality in patients with severe ARDS (PaO₂/FiO₂ < 80 mm Hg for more than 6 hours) compared to conventional mechanical ventilation (35% vs 46%) [82].

However, 28% of the conventional ventilation group eventually crossed over to ECMO, complicating conclusions about ECMO's effectiveness.

Additionally, the ARDS guidelines make two conditional recommendations for utilizing higher positive end-expiratory pressure (PEEP) and recruitment maneuvers in moderate-to-severe ARDS cases [77]. These strategies are theorized to open collapsed alveoli and enhance lung compliance and gas exchange. However, a large randomized trial found that recruitment maneuvers combined with higher PEEP titration (compared to lower PEEP care) increased 28-day mortality in ARDS patients (hazard ratio 1.20; 95% CI 1.01-1.42) [83]. A meta-analysis of 3562 ARDS patients from nine trials revealed that decreases in driving pressure (defined as tidal volume divided by respiratory system compliance or plateau pressure minus PEEP) correlated with increased survival [84]. This suggests that PEEP may benefit patients with greater lung recruitability while posing risks of overdistention in others [85-87].

Several modes of mechanical ventilation have been examined for ARDS patients. Airway pressure release ventilation (APRV) is a mode that alternates between two levels of continuous positive airway pressure, allowing spontaneous breathing during any phase of the ventilatory cycle. Early studies on APRV in ARDS patients demonstrated improvements in cardiac output, gas exchange, lung compliance, and reduced sedation and mechanical ventilation duration compared to conventional mechanical ventilation (which did not employ LPV strategies) [88,89]. In animal studies, APRV reduced lung edema and preserved key lung components, such as E-cadherin and surfactant protein, compared to low tidal volume ventilation [90]. Additionally, APRV prevented acute lung injury (ALI) in normal lungs, resulting in significantly higher $\text{PaO}_2/\text{FiO}_2$ ratios (478 vs 242, respectively, $P < .5$) [91]. A systematic review of observational trauma patient data found that early APRV use reduced ARDS incidence; however, it was unclear whether the comparison group used low tidal volume ventilation [92]. Recent studies comparing APRV with ventilation strategies targeting tidal volumes of 8 to 10 mL/kg have failed to establish a clear benefit for APRV in ARDS patients [93-95]. Nonetheless, a recent single-center, randomized controlled trial involving early APRV (initiated within 48 hours of mechanical ventilation) in 138 ARDS patients ($\text{PaO}_2/\text{FiO}_2 < 250$) found benefits in terms of ventilator-free days, extubation, tracheostomy, and ICU mortality [96]. APRV patients also required fewer proning episodes, neuromuscular blockade (NMB), and recruitment maneuvers. Importantly, the study's APRV protocol avoided high peak pressures and tidal volumes, potentially conferring greater lung protection. Given the conflicting data, there is insufficient evidence to universally recommend APRV for ARDS patients.

Inverse ratio ventilation (IRV) is an alternative mechanical ventilation strategy that has been tested in patients with ARDS. IRV alters the conventional inspiratory-to-expiratory time ratio, extending the inspiratory phase relative to the expiratory phase. This approach is theoretically aimed at increasing mean airway pressure to facilitate the recruitment of collapsed alveoli. IRV can be applied in both pressure-controlled and volume-controlled ventilation modes. However, a significant drawback of IRV is the potential for air trapping and auto-PEEP, particularly in individuals with obstructive lung conditions. Although limited

high-quality studies have evaluated IRV in ARDS, available research indicates that IRV has little effect on improving oxygenation, cardiac output, or CO₂ elimination when compared to standard ventilation methods, and may exacerbate gas exchange issues, volutrauma, and hemodynamic instability [97–100].

Given the compromised lung function in ARDS patients, they are especially susceptible to air trapping and auto-PEEP, regardless of the ventilation mode used. Esophageal manometry has been proposed as a tool for detecting auto-PEEP and guiding ventilator adjustments to reduce transpulmonary pressures, thereby enhancing gas exchange in ARDS patients [101–104]. In a randomized controlled trial involving 61 ARDS patients, Talmor et al. explored the effects of PEEP titration based on pleural pressures measured via esophageal manometry. The study found that patients whose PEEP was adjusted to achieve a transpulmonary pressure of 0 to 10 cm H₂O at end expiration exhibited improved oxygenation and lung compliance [105]. Similarly, Soroksy et al. used esophageal manometry to adjust tidal volume in ARDS patients, successfully treating severe hypercapnia [106]. However, Chiumello et al. determined that PEEP titration using esophageal manometry did not consistently predict lung recruitment, as assessed by computed tomography scans [107]. A more recent study found no significant difference in mortality or ventilator-free days between ARDS patients who received PEEP titration based on esophageal pressure measurements and those following a high PEEP-FiO₂ strategy [108]. As such, further robust studies are needed before esophageal manometry can be considered a standard practice in ARDS management. A large-scale randomized trial, the Esophageal Pressure- Guided Ventilation 2 study, is currently being conducted to evaluate the clinical impact of esophageal manometry in ARDS patients on outcomes such as mortality and ventilator-free days [109].

Positioning For patients with severe ARDS (PaO₂/FiO₂ ratio <100), prone positioning for a minimum of 12 hours per day is recommended. The PROSEVA trial demonstrated that prone positioning in these patients reduced 28-day mortality by over 50% [110]. While prone positioning represents a significant shift in practice for many intensive care units, it poses logistical challenges and may increase risks, including accidental displacement of endotracheal tubes, the need for deeper sedation, limited opportunities for early mobilization, and a higher incidence of pressure ulcers.

Nutritional Management Patients with ARDS exhibit a highly catabolic state, necessitating adequate nutritional support to counterbalance caloric and protein losses without exacerbating fluid overload [111]. The EDEN trial explored the optimal nutritional approach by comparing the outcomes of patients receiving trophic enteral feeds (20 kcal/h) versus full enteral feeds (25–30 kcal/kg/day of nonprotein calories and 1.2–1.6 g/kg/day of protein) during the first six days of mechanical ventilation. After this period, both groups aimed for full enteral nutrition [112]. No significant differences were observed in terms of ventilator-free days, short- or long-term mortality, physical function, or secondary complications between the two feeding strategies [113,114]. However, the full enteral feeds group experienced higher rates of gastrointestinal complications, such as vomiting, elevated gastric residuals, hyperglycemia, and constipation [112].

The optimal route for delivering nutrition to ARDS patients remains uncertain. Concerns have been raised about parenteral nutrition, particularly the infusion of large quantities of intravenous (IV) fat emulsions, potentially exacerbating alveolar epithelial inflammation. Lekka et al. compared the effects of lipid-containing total parenteral nutrition to placebo in ARDS patients, finding that lipid administration led to worsening oxygenation, reduced pulmonary compliance, and increased pulmonary vascular resistance [115]. Similarly, Suchner et al. reported that rapid IV fat infusion over six hours worsened oxygenation compared to slower 24-hour infusions [116]. Consequently, IV lipid infusions may be detrimental to ARDS patients. However, the CALORIES Trial by Harvey et al., which involved 2400 critically ill patients (including those with ARDS), found no significant differences in 30- and 90-day mortality or infection rates between those receiving full parenteral versus enteral nutrition [117]. Further research is necessary to determine the most effective nutritional delivery method for critically ill ARDS patients.

The literature presents mixed findings regarding the clinical effectiveness of nutritional antioxidant supplementation for reducing pulmonary inflammation in ARDS patients. Two separate studies (sample sizes of 146 and 100, respectively) assessed the impact of eicosapentaenoic acid, gamma-linolenic acid, and antioxidants—key components of an immune-modulating diet—on patients with ARDS. Both studies reported enhanced oxygenation and reduced time on mechanical ventilation in those receiving the immune-modulating supplements [118,119]. However, a more recent trial by Rice et al., which administered ω -3 fatty acids, γ -linolenic acid, and antioxidants to ARDS patients, was terminated early due to lack of clinical benefit [120]. Similarly, Stapleton and colleagues conducted a study involving 90 ARDS patients comparing fish oil supplementation to placebo, finding no significant differences in pulmonary biomarkers or clinical outcomes [121]. Given the small sample sizes and contradictory results of these studies, further research is required to evaluate the efficacy of immune-modulating diets in ARDS patients. Continuous Positive Airway Pressure (CPAP) and Bilevel Positive Airway Pressure (BiPAP) are two forms of non-invasive ventilation that have been utilized in the management of Acute Respiratory Distress Syndrome (ARDS). Both methods aim to improve oxygenation and reduce the work of breathing in patients experiencing respiratory failure. Here's an overview of each modality's role in ARDS:

Continuous Positive Airway Pressure (CPAP)

- **Mechanism:** CPAP delivers a constant level of positive airway pressure throughout the entire respiratory cycle, which helps maintain airway patency and prevents the collapse of alveoli. This mode increases functional residual capacity (FRC) and improves oxygenation by recruiting collapsed alveoli.
- **Indications:** CPAP is typically used in patients with mild to moderate ARDS who can tolerate it and do not require intubation. It can be beneficial in patients with hypoxemia and increased work of breathing.
- **Benefits:**
 - Enhances oxygenation and decreases the need for intubation in selected patients.

- Reduces the work of breathing and may alleviate respiratory fatigue.
- Can be applied in a variety of settings, including at home.
- **Limitations:**
 - CPAP may not be suitable for patients with significant respiratory acidosis or those who are unable to cooperate with the treatment.
 - There is a risk of discomfort, mask leaks, and increased work of breathing if not properly fitted.

Bilevel Positive Airway Pressure (BiPAP)

- **Mechanism:** BiPAP provides two levels of pressure: a higher inspiratory pressure (IPAP) during inhalation and a lower expiratory pressure (EPAP) during exhalation. This dual pressure support helps facilitate ventilation while maintaining airway patency.
- **Indications:** BiPAP is often indicated for patients with more severe ARDS or those showing signs of respiratory acidosis. It is particularly useful in patients who require assistance with ventilation but are not ready for intubation.
- **Benefits:**
 - Improves ventilation and gas exchange by allowing for a higher inspiratory pressure, which can enhance tidal volume.
 - Helps reduce the work of breathing and can be more comfortable for patients compared to CPAP.
 - Decreases the risk of respiratory muscle fatigue and may improve pH levels in patients with respiratory acidosis.
- **Limitations:**
 - Similar to CPAP, BiPAP may not be appropriate for patients who are uncooperative or unable to protect their airway.
 - It may be associated with increased aspiration risk in some patients.

Both CPAP and BiPAP can be effective non-invasive ventilation strategies in the management of ARDS, especially for patients who are not candidates for intubation. The choice between CPAP and BiPAP largely depends on the severity of respiratory failure, the patient's ability to cooperate with treatment, and specific clinical circumstances. While non-invasive ventilation can improve oxygenation and decrease the need for intubation, close monitoring and careful patient selection are essential to optimize outcomes in ARDS management.

Conclusion

Acute Respiratory Distress Syndrome (ARDS) is a critical condition characterized by acute hypoxic respiratory failure and various underlying conditions. Identified by Ashbaugh et al. in the 1960s, ARDS significantly affects morbidity and mortality rates globally, leading to approximately 75,000 deaths annually in the U.S. and impacting around 3 million individuals worldwide. Despite improvements in mortality rates due to advancements in critical care, ARDS remains a significant concern in emergency and critical settings. The aim of the study was to evaluate the prehospital use of Continuous Positive Airway Pressure (CPAP) and Bilevel Positive Airway Pressure (BiPAP) in managing respiratory

distress among ARDS patients. Both interventions offer non-invasive alternatives to mechanical ventilation and have shown promise in enhancing oxygenation and reducing intubation rates in critically ill patients. Through a comprehensive review of existing literature, the efficacy of these non-invasive ventilation strategies was assessed, emphasizing their role in emergency medical services (EMS). The findings suggest that CPAP and BiPAP significantly improve patient outcomes by stabilizing conditions during transport, reducing respiratory distress, and providing comfort. The studies analyzed indicate that implementing these strategies can lower the need for invasive ventilation methods, thereby minimizing the associated risks and complications. In conclusion, the prehospital application of CPAP and BiPAP is a valuable strategy in the EMS management of ARDS. Their integration into practice can potentially improve survival rates and overall patient outcomes in respiratory distress scenarios. Ongoing education and protocol development are essential to enhance the effectiveness of these interventions within the EMS framework. Further research is warranted to establish standardized guidelines for the use of non-invasive ventilation in prehospital settings, ensuring timely and effective management of ARDS and related respiratory conditions.

References

1. Ashbaugh D, Bigelow DB, Petty T, Levine B. Acute respiratory distress in adults. *Lancet*. 1967;290(7511):319–323.
2. Luhr OR, Antonsen K, Karlsson M, et al. Incidence and mortality after acute respiratory failure and acute respiratory distress syndrome in Sweden, Denmark, and Iceland. *Am J Respir Crit Care Med*. 1999;159(6):1849–1861.
3. Villar J. What is the acute respiratory distress syndrome? *Respir Care*. 2011;56(10):1539–1545.
4. Bersten AD, Edibam C, Hunt T, et al. Incidence and mortality of acute lung injury and the acute respiratory distress syndrome in three Australian States. *Am J Respir Crit Care Med*. 2002;165(4):443–448.
5. Stapleton RD, Wang BM, Hudson LD, Rubenfeld GD, Caldwell ES, Steinberg KP. Causes and timing of death in patients with ARDS. *Chest*. 2005;128(2):525–532.
6. Erickson SE, Martin GS, Davis JL, Matthay MA, Eisner MD, Network NNA. Recent trends in acute lung injury mortality: 1996–2005. *Crit Care Med*. 2009;37(5):1574–1579.
7. Rubenfeld GD, Caldwell E, Peabody E, et al. Incidence and outcomes of acute lung injury. *N Engl J Med*. 2005;353(16):1685–1693.
8. Bellani G, Laffey JG, Pham T, et al. Epidemiology, patterns of care, and mortality for patients with acute respiratory distress syndrome in intensive care units in 50 countries. *JAMA*. 2016;315(8):788–800.
9. Bernard GR, Artigas A, Brigham KL, et al. The American-European Consensus Conference on ARDS. Definitions, mechanisms, relevant outcomes, and clinical trial coordination. *Am J Respir Crit Care Med*. 1994;149(3 pt 1):818–824.

10. Ranieri VM, Rubenfeld GD, Thompson BT et al; ARDS Definition Task Force. Acute respiratory distress syndrome: the Berlin definition. *JAMA*. 2012;307(23):2526–2533.
11. Ferguson ND, Fan E, Camporota L, et al. The Berlin definition of ARDS: an expanded rationale, justification, and supplementary material. *Intensive Care Med*. 2012;38(10):1573–1582.
12. Katzenstein AL, Bloor CM, Leibow AA. Diffuse alveolar damage – the role of oxygen, shock, and related factors. A review. *Am J Pathol*. 1976;85(1):209–228.
13. Piantadosi CA, Schwartz DA. The acute respiratory distress syndrome. *Ann Intern Med*. 2004;141(6):460–470.
14. Anderson WR, Thielen K. Correlative study of adult respiratory distress syndrome by light, scanning, and transmission electron microscopy. *Ultrastruct Pathol*. 1992;16(6):615–628.
15. Pratt PC, Vollmer RT, Shelburne JD, Crapo JD. Pulmonary morphology in a multihospital collaborative extracorporeal membrane oxygenation project. I. Light microscopy. *Am J Pathol*. 1979;95(1):191–214.
16. Bachofen M, Weibel ER. Alterations of the gas exchange apparatus in adult respiratory insufficiency associated with septicemia. *Am Rev Respir Dis*. 1977;116(4):589–615.
17. Pierrakos C, Karanikolas M, Scolletta S, Karamouzos V, Velissaris D. Acute respiratory distress syndrome: pathophysiology and therapeutic options. *J Clin Med Res*. 2012;4(1):7–16.
18. de Hemptinne Q, Remmelink M, Brimioule S, Salmon I, Vincent JL. ARDS: a clinicopathological confrontation. *Chest*. 2009;135(4):944–949.
19. Sarmiento X, Guardiola JJ, Almirall J, et al. Discrepancy between clinical criteria for diagnosing acute respiratory distress syndrome secondary to community acquired pneumonia with autopsy findings of diffuse alveolar damage. *Respir Med*. 2011;105(8):1170–1175.
20. Esteban A, Fernández-Segoviano P, Frutos-Vivar F, et al. Comparison of clinical criteria for the acute respiratory distress syndrome with autopsy findings. *Ann Intern Med*. 2004;141(6):440–445.
21. Pinheiro BV, Muraoka FS, Assis RVC, et al. Accuracy of clinical diagnosis of acute respiratory distress syndrome in comparison with autopsy findings. *J Bras Pneumol*. 2007;33(4):423–428.
22. Kao KC, Hu HC, Chang CH, et al. Diffuse alveolar damage associated mortality in selected acute respiratory distress syndrome patients with open lung biopsy. *Crit Care*. 2015;19(1):228.
23. Thompson BT, Michael AM. The Berlin definition of ARDS versus pathological evidence of diffuse alveolar damage. *Am J Respir crit Care Med*. 2013;187(7):675–677.

24. Hudson LD, Milberg JA, Anardi D, Mauder RJ. Clinical risks for development of the acute respiratory distress syndrome. *Am J Respir crit Care Med.* 1995;151(2 pt 1):293–301.
25. Pepe PE, Potkin RT, Reus DH, Hudson LD, Carrico CJ. Clinical predictors of the adult respiratory distress syndrome. *Am J Surg.* 1982;144(1):124–130.
26. Eworuke E, Major JM, Gilbert McClain LI. National incidence rates for acute respiratory distress syndrome (ARDS) and ARDS cause-specific factors in the United States (2006-2014). *J Crit Care.* 2018;47:192–197.
27. Marshall RP, Webb S, Hill MR, Humphries SE, Laurent GJ. Genetic polymorphisms associated with susceptibility and outcome in ARDS. *Chest.* 2002;121(3 suppl):68S–69S.
28. Marshall RP, Webb S, Bellingan GJ, et al. Angiotensin converting enzyme insertion/deletion polymorphism is associated with susceptibility and outcome in acute respiratory distress syndrome. *Am J Respir crit Care Med.* 2002;166(5):646–650.
29. Copland IB, Kavanagh BP, Engelberts D, McKerlie C, Belik J, Post M. Early changes in lung gene expression due to high tidal volume. *Am J Respir crit Care Med.* 2003;168(9):1051–1059.
30. Grigoryev DN, Finigan JH, Hassoun P, Garcia JGN. Science review: searching for gene candidates in acute lung injury. *Crit Care.* 2004;8(6):440.
31. Mikkelsen ME, Shah CV, Meyer NJ, et al. The epidemiology of acute respiratory distress syndrome in patients presenting to the emergency department with severe sepsis. *Shock.* 2013;40(5):375–381.
32. Gajic O, Dabbagh O, Park PK, et al. Early identification of patients at risk of acute lung injury: evaluation of lung injury prediction score in a multicenter cohort study. *Am J Respir crit Care Med.* 2011;183(4):462–470.
33. Ferguson ND, Frutos-Vivar F, Esteban A, et al. Clinical risk conditions for acute lung injury in the intensive care unit and hospital ward: a prospective observational study. *Crit Care.* 2007;11(5):R96.
34. Fein AM, Calalang-Colucci MG. Acute lung injury and acute respiratory distress syndrome in sepsis and septic shock. *Crit Care Clin.* 2000;16(2):289–317.
35. Pelosi P, D'Onofrio D, Chiumello D, et al. Pulmonary and extrapulmonary acute respiratory distress syndrome are different. *Eur Respir J Suppl.* 2003;42:48s–56s.
36. Sheu CC, Gong MN, Zhai R, et al. The influence of infection sites on development and mortality of ARDS. *Intensive Care Med.* 2010;36(6):963–970.
37. Kojicic M, Li G, Hanson AC, et al. Risk factors for the development of acute lung injury in patients with infectious pneumonia. *Crit Care.* 2012;16(2):R46.
38. Lee A, Festic E, Park PK, et al. Characteristics and outcomes of patients hospitalized following pulmonary aspiration. *Chest.* 2014;146(4):899–907.
39. Treggiani MM, Hudson LD, Martin DP, Weiss NS, Caldwell E, Rubenfeld G. Effect of acute lung injury and acute respiratory distress syndrome on outcome in critically ill trauma patients. *Crit Care Med.* 2004;32(2):327–331.

40. Calfee CS, Eisner MD, Ware LB, et al. Trauma-associated lung injury differs clinically and biologically from acute lung injury due to other clinical disorders. *Crit Care Med.* 2007;35(10):2243–2250.
41. Moss M, Gillespie MK, Ackerson L, Moore FA, Moore EE, Parsons PE. Endothelial cell activity varies in patients at risk for the adult respiratory distress syndrome. *Crit Care Med.* 1996;24(11):1782–1786.
42. Toy P, Popovsky MA, Abraham E, et al. Transfusion-related acute lung injury: definition and review. *Crit Care Med.* 2005;33(4):721–726.
43. Zilberberg MD, Carter C, Lefebvre P, et al. Red blood cell transfusions and the risk of acute respiratory distress syndrome among the critically ill: a cohort study. *Crit Care.* 2007;11(3):R63.
44. Khan H, Belsher J, Yilmaz M, et al. Fresh-frozen plasma and platelet transfusions are associated with development of acute lung injury in critically ill medical patients. *Chest.* 2007;131(5):1308–1314.
45. Levitt JE, Calfee CS, Goldstein BA, Vojnik R, Matthay MA. Early acute lung injury: criteria for identifying lung injury prior to the need for positive pressure ventilation*. *Crit Care Med.* 2013;41(8):1929–1937.
46. Crader KM, Repine DJJ, Repine JE. Breath biomarkers and the acute respiratory distress syndrome. *J Pulm Respir Med.* 2012;2(1):1–9.
47. Donnelly SC, Strieter RM, Kunkel SL, et al. Interleukin-8 and development of adult respiratory distress syndrome in at-risk patient groups. *Lancet.* 1993;341(8846):643–647.
48. Villar J, Pérez-Méndez L, Espinosa E, et al. Serum lipopolysaccharide binding protein levels predict severity of lung injury and mortality in patients with severe sepsis. *PLoS One.* 2009;4(8):e6818.
49. Agrawal A, Matthay MA, Kangelaris KN, et al. Plasma angiopoietin-2 predicts the onset of acute lung injury in critically ill patients. *Am J Respir Crit Care Med.* 2013;187(7):736–742.
50. Terpstra ML, Aman J, van Nieuw Amerongen GP, Groeneveld ABJ. Plasma biomarkers for acute respiratory distress syndrome: a systematic review and meta-analysis*. *Crit Care Med.* 2014;42(3):691–700.
51. Calfee CS, Ware LB, Glidden DV, et al. Use of risk reclassification with multiple biomarkers improves mortality prediction in acute lung injury. *Crit Care Med.* 2011;39(4):711–717.
52. Ware LB, Koyama T, Billheimer DD, et al. Prognostic and pathogenetic value of combining clinical and biochemical indices in patients with acute lung injury. *Chest.* 2010;137(2):288–296.
53. Iscimen R, Cartin-Ceba R, Yilmaz M, et al. Risk factors for the development of acute lung injury in patients with septic shock: an observational cohort study. *Crit Care Med.* 2008;36(5):1518–1522.
54. Jia X, Malhotra A, Saeed M, Mark RG, Talmor D. Risk factors for ARDS in patients receiving mechanical ventilation for greater than 48 hours. *Chest.* 2008;133(4):853–861.

55. Yao S, Mao T, Fang W, Xu M, Chen W. Incidence and risk factors for acute lung injury after open thoracotomy for thoracic diseases. *J Thorac Dis.* 2013;5(4):455–460.
56. Evans RG, Naidu B. Does a conservative fluid management strategy in the perioperative management of lung resection patients reduce the risk of acute lung injury? *Interact Cardiovasc Thorac Surg.* 2012;15(3):498–504.
57. Hughes CG, Weavind L, Banerjee A, Mercaldo ND, Schildcrout JS, Pandharipande PP. Intraoperative risk factors for acute respiratory distress syndrome in critically ill patients. *Anesth Analg.* 2010;111(2):464–467.
58. Wiedemann HP, Wheeler AP, Bernard GR, et al. Comparison of two fluid-management strategies in acute lung injury. *N Engl J Med.* 2006;354(24):2564–2575.
59. Rosenberg AL, Dechert RE, Park PK, Bartlett RH, Network NNA. Review of a large clinical series: association of cumulative fluid balance on outcome in acute lung injury: a retrospective review of the ARDSnet tidal volume study cohort. *J Intensive Care Med.* 2009;24(1):35–46.
60. Seeley EJ. Fluid therapy during acute respiratory distress syndrome: less is more, simplified*. *Crit Care Med.* 2015;43(2):477–478.
61. Gong MN, Thompson BT, Williams P, Pothier L, Boyce PD, Christiani DC. Clinical predictors of and mortality in acute respiratory distress syndrome: potential role of red cell transfusion. *Crit Care Med.* 2005;33(6):1191–1198.
62. Hébert PC, Wells G, Blajchman MA, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. Transfusion requirements in critical care investigators, canadian critical care trials group. *N Engl J Med.* 1999;340(6):409–417.
63. Park PK, Cannon JW, Ye W, et al. Transfusion strategies and development of acute respiratory distress syndrome in combat casualty care. *J Trauma Acute Care Surg.* 2013;75(2 suppl 2):S238–S246.
64. Popovsky MA, Moore SB. Diagnostic and pathogenetic considerations in transfusion-related acute lung injury. *Transfusion.* 1985;25(6):573–577.
65. Curtis BR, McFarland JG. Mechanisms of transfusion-related acute lung injury (TRALI): anti-leukocyte antibodies. *Critical care medicine.* 2006;34(5 suppl):S118–S123.
66. Vlaar AP, Juffermans NP. Transfusion-related acute lung injury: a clinical review. *Lancet.* 2013;382(9896):984–994.
67. Toy P, Gajic O, Bacchetti P, et al. Transfusion-related acute lung injury: incidence and risk factors. *Blood.* 2012;119(7):1757–1767.
68. Gajic O, Rana R, Winters JL, et al. Transfusion-related acute lung injury in the critically ill: prospective nested case-control study. *Am J Respir Crit Care Med.* 2007;176(9):886–891.
69. Price TH. *Standards for Blood Banks and Transfusion Services.* Bethesda, MD: AABB; 2008.
70. Eder AF, Herron R, Strupp A, et al. Transfusion-related acute lung injury surveillance (2003-2005) and the potential impact of the selective use of

plasma from male donors in the American Red Cross. *Transfusion*. 2007;47(4):599–607.

71. Silliman CC, Thurman GW, Ambruso DR. Stored blood components contain agents that prime the neutrophil NADPH oxidase through the platelet-activating-factor receptor. *Vox Sanguinis*. 1992;63(2):133–136.
72. Tung JP, Fraser JF, Nataatmadja M, et al. Age of blood and recipient factors determine the severity of transfusion-related acute lung injury (TRALI). *Crit Care*. 2012;16(1):R19.
73. Vlaar APJ, Binnekade JM, Prins D, et al. Risk factors and outcome of transfusion-related acute lung injury in the critically ill: a nested case-control study. *Crit Care Med*. 2010;38(3):771–778.
74. Middelburg RA, Borkent-Raven BA, Borkent B, et al. Storage time of blood products and transfusion-related acute lung injury. *Transfusion*. 2012;52(3):658–667.
75. Kor DJ, Kashyap R, Weiskopf RB, et al. Fresh red blood cell transfusion and short-term pulmonary, immunologic, and coagulation status: a randomized clinical trial. *Am J Respir Crit Care Med*. 2012;185(8):842–850.
76. Fan E, Brodie D, Slutsky AS. Acute respiratory distress syndrome: advances in diagnosis and treatment. *JAMA*. 2018;319(7):698–710.
77. Howell MD, Davis AM. Management of ARDS in adults. *JAMA*. 2018;319(7):711–712.
78. Fan E, Del Sorbo L, Goligher EC, et al. An official American Thoracic Society/European Society of Intensive Care Medicine/Society of Critical Care Medicine clinical practice guideline: mechanical ventilation in adult patients with acute respiratory distress syndrome. *Am J Respir Crit Care Med*. 2017;195(9):1253–1263.
79. Ferguson ND, Cook DJ, Guyatt GH, et al. High-frequency oscillation in early acute respiratory distress syndrome. *N Engl J Med*. 2013;368(9):795–805.
80. Lall R, Hamilton P, Young D, et al. A randomised controlled trial and cost-effectiveness analysis of high-frequency oscillatory ventilation against conventional artificial ventilation for adults with acute respiratory distress syndrome. The OSCAR (OSCillation in ARDS) study. *Health Technol Assess*. 2015;19(23):1–177, vii.
81. Sud S, Sud M, Friedrich JO, et al. High-frequency oscillatory ventilation versus conventional ventilation for acute respiratory distress syndrome. *Cochrane Database Syst Rev*. 2016;(4):CD004085.
82. Combes A, Hajage D, Capellier G, et al. Extracorporeal membrane oxygenation for severe acute respiratory distress syndrome. *N Engl J Med*. 2018;378(21):1965–1975.
83. Cavalcanti AB, Suzumura EA, Laranjeira LN, et al. Effect of lung recruitment and titrated positive end-expiratory pressure (PEEP) vs low PEEP on mortality in patients with acute respiratory distress syndrome: a randomized clinical trial. *JAMA*. 2017;318(14):1335–1345.

84. Amato MB, Meade MO, Slutsky AS, et al. Driving pressure and survival in the acute respiratory distress syndrome. *N Engl J Med.* 2015;372(8):747–755.
85. Grasso S, Fanelli V, Cafarelli A, et al. Effects of high versus low positive end-expiratory pressures in acute respiratory distress syndrome. *Am J Respir Crit Care Med.* 2005;171(9):1002–1008.
86. Vieira SR, Puybasset L, Lu Q, et al. A scanographic assessment of pulmonary morphology in acute lung injury. Significance of the lower inflection point detected on the lung pressure-volume curve. *Am J Respir Crit Care Med.* 1999;159(5 pt 1):1612–1623.
87. Gattinoni L, Caironi P, Cressoni M, et al. Lung recruitment in patients with the acute respiratory distress syndrome. *N Engl J Med.* 2006;354(17):1775–1786.
88. Putensen C, Mutz NJ, Putensen-Himmer G, Zinserling J. Spontaneous breathing during ventilatory support improves ventilation-perfusion distributions in patients with acute respiratory distress syndrome. *Am J Respir Crit Care Med.* 1999;159(4 pt 1):1241–1248.
89. Putensen C, Zech S, Wrigge H, et al. Long-term effects of spontaneous breathing during ventilatory support in patients with acute lung injury. *Am J Respir Crit Care Med.* 2001;164(1):43–49.
90. Roy S, Habashi N, Sadowitz B, et al. Early airway pressure release ventilation prevents ARDS – a novel preventive approach to lung injury. *Shock.* 2013;39(1):28.
91. Emr B, Gatto LA, Roy S, et al. Airway pressure release ventilation prevents ventilator-induced lung injury in normal lungs. *JAMA Surg.* 2013;148(11):1005–1012.
92. Andrews PL, Shiber JR, Jaruga-Killeen E, et al. Early application of airway pressure release ventilation may reduce mortality in high-risk trauma patients: a systematic review of observational trauma ARDS literature. *J Trauma Acute Care Surg.* 2013;75(4):635–641.
93. Varpula T, Valta P, Niemi R, Takkunen O, Hynynen M, Pettila VV. Airway pressure release ventilation as a primary ventilatory mode in acute respiratory distress syndrome. *Acta Anaesthesiol Scand.* 2004;48(6):722–731.
94. Maxwell RA, Green JM, Waldrop J, et al. A randomized prospective trial of airway pressure release ventilation and low tidal volume ventilation in adult trauma patients with acute respiratory failure. *J Trauma.* 2010;69(3):501–510; discussion 511.
95. Gonzalez M, Arroliga AC, Frutos-Vivar F, et al. Airway pressure release ventilation versus assist-control ventilation: a comparative propensity score and international cohort study. *Intensive Care Med.* 2010;36(5):817–827.
96. Zhou Y, Jin X, Lv Y, et al. Early application of airway pressure release ventilation may reduce the duration of mechanical ventilation in acute respiratory distress syndrome. *2017;43(11):1648–1659.*

97. Mercat A, Titiriga M, Anguel N, Richard C, Teboul JL. Inverse ratio ventilation (I/E = 2/1) in acute respiratory distress syndrome: a six-hour controlled study. *Am J Respir Crit Care Med.* 1997;155(5):1637–1642.
98. Lessard MR, Guerot E, Lorino H, Lemaire F, Brochard L. Effects of pressure-controlled with different I:E ratios versus volume-controlled ventilation on respiratory mechanics, gas exchange, and hemodynamics in patients with adult respiratory distress syndrome. *Anesthesiology.* 1994;80(5):983–991.
99. Mancebo J, Vallverdu I, Bak E, et al. Volume-controlled ventilation and pressure-controlled inverse ratio ventilation: a comparison of their effects in ARDS patients. *Monaldi Arch Chest Dis* 1994;49(3):201–207.
100. Huang CC, Shih MJ, Tsai YH, Chang YC, Tsao TC, Hsu KH. Effects of inverse ratio ventilation versus positive end-expiratory pressure on gas exchange and gastric intramucosal PCO₂ and pH under constant mean airway pressure in acute respiratory distress syndrome. *Anesthesiology.* 2001;95(5):1182–1188.
101. Soroksky A, Esquinas A. Goal-directed mechanical ventilation: are we aiming at the right goals? A proposal for an alternative approach aiming at optimal lung compliance, guided by esophageal pressure in acute respiratory failure. *Crit Care Res Pract.* 2012. doi:10.1155/2012/597932.
102. Chiumello D, Cressoni M, Colombo A, et al. The assessment of transpulmonary pressure in mechanically ventilated ARDS patients. *Intensive Care Med.* 2014;40(11):1670–1678.
103. Chiumello D, Guerin C. Understanding the setting of PEEP from esophageal pressure in patients with ARDS. *Intensive Care Med.* 2015;41(8):1465–1467.
104. Talmor D, Sarge T, O'Donnell CR, et al. Esophageal and transpulmonary pressures in acute respiratory failure. *Crit Care Med.* 2006;34(5):1389–1394.
105. Talmor D, Sarge T, Malhotra A, et al. Mechanical ventilation guided by esophageal pressure in acute lung injury. *N Engl J Med.* 2008;359(20):2095–2104.
106. Soroksky A, Kheifets J, Girsh Solomonovich Z, Tayem E, Gingy Ronen B, Rozhavsky B. Managing hypercapnia in patients with severe ARDS and low respiratory system compliance: the role of esophageal pressure monitoring – a case-cohort study. *Bio Med Res Int.* 2015;2015:385042.
107. Chiumello D, Cressoni M, Carlesso E, et al. Bedside selection of positive end-expiratory pressure in mild, moderate, and severe acute respiratory distress syndrome. *Crit Care Med.* 2014;42(2):252–264.
108. Beitzler JR, Sarge T, Banner-Goodspeed VM, et al. Effect of titrating positive end-expiratory pressure (PEEP) with an esophageal pressure-guided strategy vs an empirical high PEEP-FiO₂ strategy on death and days free from mechanical ventilation among patients with acute respiratory distress syndrome: a randomized clinical trial. *JAMA.* 2019;321(9):846–857.

109. Fish E, Novack V, Banner-Goodspeed VM, Sarge T, Loring S, Talmor D. The esophageal pressure-guided ventilation 2 (EPVent2) trial protocol: a multicentre, randomised clinical trial of mechanical ventilation guided by transpulmonary pressure. *BMJ Open*. 2014;4(9):e006356.
110. Guerin C, Reignier J, Richard JC, et al. Prone positioning in severe acute respiratory distress syndrome. *N Engl J Med*. 2013;368(23):2159–2168.
111. Martindale RG, McClave SA, Vanek VW, et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: society of critical care medicine and american society for parenteral and enteral nutrition: executive summary. *Crit Care Med*. 2009;37(5):1757–1761.
112. Rice TW, Wheeler AP, Thompson BT, et al. Initial trophic vs full enteral feeding in patients with acute lung injury: the EDEN randomized trial. *JAMA*. 2012;307(8):795–803.
113. Needham DM, Dinglas VD, Bienvenu OJ, et al. One year outcomes in patients with acute lung injury randomised to initial trophic or full enteral feeding: prospective follow-up of EDEN randomised trial. *BMJ*. 2013;346:f1532.
114. Needham DM, Dinglas VD, Morris PE, et al. Physical and cognitive performance of patients with acute lung injury 1 year after initial trophic versus full enteral feeding. EDEN trial follow-up. *Am J Respir Crit Care Med*. 2013;188(5):567–576.
115. Lekka ME, Liokatis S, Nathanael C, Galani V, Nakos G. The impact of intravenous fat emulsion administration in acute lung injury. *Am J Respir Crit Care Med*. 2004;169(5):638–644.
116. Suchner U, Katz DP, Furst P, et al. Effects of intravenous fat emulsions on lung function in patients with acute respiratory distress syndrome or sepsis. *Crit Care Med*. 2001;29(8):1569–1574.
117. Harvey SE, Parrott F, Harrison DA, et al. Trial of the route of early nutritional support in critically ill adults. *N Engl J Med*. 2014;371(18):1673–1684.
118. Singer P, Theilla M, Fisher H, Gibstein L, Grozovski E, Cohen J. Benefit of an enteral diet enriched with eicosapentaenoic acid and gamma-linolenic acid in ventilated patients with acute lung injury. *Crit Care Med*. 2006;34(4):1033–1038.
119. Gadek JE, DeMichele SJ, Karlstad MD, et al. Effect of enteral feeding with eicosapentaenoic acid, gamma-linolenic acid, and antioxidants in patients with acute respiratory distress syndrome. Enteral Nutrition in ARDS Study Group. *Crit Care Med*. 1999;27(8):1409–1420.
120. Rice TW, Wheeler AP, Thompson BT, et al. Enteral omega-3 fatty acid, gamma-linolenic acid, and antioxidant supplementation in acute lunginjury. *JAMA*. 2011;306(14):1574–1581.

121. Stapleton RD, Martin TR, Weiss NS, et al. A phase II randomized placebo-controlled trial of omega-3 fatty acids for the treatment of acute lung injury. *Crit Care Med.* 2011;39(7):1655–1662.
122. Devlin JW, Skrobik Y, Gélinas C, et al. Clinical Practice Guidelines for the Prevention and Management of pain, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU. *Crit Care Med.* 2018;46(9):e825–e873.

ادارة طوارئ وخدمة الاسعاف لحالات ضيق التنفس: استخدام BiPAP و CPAP في مرحلة ما قبل المستشفى

الملخص:

الخلفية: تم التعرف على متلازمة الضائقة التنفسية الحادة (ARDS) لأول مرة في السبعينيات، وهي تظهر كفشل تنفسى حاد نتيجة نقص الأكسجين بسبب أسباب متنوعة مثل العووى والإصابات. تتفاوت نسبة حدوثها عالمياً، حيث تؤثر على 7.2 إلى 34 لكل 100,000 سنة-شخص. في حين كانت نسبة الوفيات التاريخية المرتبطة بـ ARDS حوالي 60%， فقد أدت التقدمات في رعاية الحالات الحرجة إلى تقليلها إلى 35-36%. على الرغم من التحسينات، فإن ARDS تسبب حوالي 75,000 حالة وفاة سنوياً في الولايات المتحدة و 3 ملايين حالة عالمياً، مما يساهم بشكل كبير في إدخال المرضى إلى وحدات العناية المركزة واحتياجات التهوية الميكانيكية.

الهدف: يهدف هذا المقال إلى استكشاف إدارة حالات ضيق التنفس في مرضي ARDS في مرحلة ما قبل المستشفى، مع التركيز على فعالية ضغط مجرى الهواء الإيجابي المستمر (CPAP) وضغط مجرى الهواء الإيجابي ثانى المستوى (BiPAP) في سياق خدمات الطوارئ الطبية (EMS). كما ترکز المراجعة أيضاً على الصورة الشعاعية لـ ARDS.

الطرق: تم إجراء مراجعة شاملة للأدبيات الموجودة، حيث تم تحليل الدراسات المتعلقة بتطبيق CPAP و BiPAP في إدارة ARDS في بيئات ما قبل المستشفى. تشمل المراجعة الفعالية والنتائج السريرية والنتائج السريرية وسلامة استراتيجيات التهوية غير الغازية هذه.

النتائج: تشير الأدلة إلى أن كل من CPAP و BiPAP مفيدين في تحسين الأكسجة وتقليل الحاجة إلى التهبيب في مرضي ARDS. هذه التدخلات تعزز أيضاً راحة المرضى ويمكن أن تثبت الحالات أثناء النقل إلى الرعاية النهائية.

الخاتمة: بعد استخدام BiPAP و CPAP في مرحلة ما قبل المستشفى نهجاً واعداً لإدارة ضيق التنفس في ARDS. إن دمج هذه الطرق غير الغازية للتهوية يمكن أن يقلل من المراضاة والوفيات، مما يبرز الحاجة إلى مزيد من التدريب والبروتوكولات في EMS.

الكلمات المفتاحية: ARDS، BiPAP، CPAP، ضيق التنفس، خدمات الطوارئ الطبية، التهوية غير الغازية.